## Correspondence

## Prolonged exclusive breast feeding and heredity as determinants in infantile atopy

Sir,

The observation in the Finnish study that prolonged breast feeding is associated with a higher incidence of infantile atopy, heredity being its only reliable predictor, is not surprising and warrants some additional considerations. I Dr David's commentary omits some of the basic biological aspects.

In many studies atopy is assessed by the onset during infancy of certain clinical signs possibly caused by atopy. Whether breast feeding can or cannot delay the onset of these signs is not the same question as whether or not atopic disease itself can be prevented. In a number of inherited metabolic disorders—phenylketonuria, for example—a suitable diet can prevent symptoms from developing, but cannot eliminate the underlying congenital disorder.

Furthermore, we are a long way from knowing whether and to what extent atopic signs and symptoms during infancy are antigen or host dependent. From findings in animals and in cultured fetal intestinal tissues, various factors available in breast milk have been shown to stimulate the gut surface, possibly exaggerating the infant's immune responses, as recently reported by Raloff.<sup>2</sup> In addition to heredity factors, breast feeding could even enhance the host's capability of eliciting a reaginic response with consequent earlier clinical presentation of atopy, an argument invoked to discourage the combination of breast and formula feeding.<sup>2</sup>

Some foods ingested by the mother pass into the milk and can sensitise the infant. Also, various milk proteins from the mother may have immunogenic power due to the frequent occurrence of polymorphic variants genetically arranged in different structural forms. They include the not strictly alimental milk proteins like immunoglobulins, blood group, and HLA specific glycoproteins and transferrin, and also specific milk proteins, like casein.<sup>3</sup> The traditional statement that mother's milk proteins are homologous for her infant is contradicted by the presence of allologous proteins which have potential immunogenic capacity when they come into contact with the immune system of an infant differing from the mother for that allotype.

It is not surprising that prolonged breast feeding may be associated with a higher incidence of infantile atopy when compared with limited breast feeding because of the more prolonged exposition to maternal allotypes and possibly because of its adjuvant effect in the sensitisation process. The same explanation might apply to the observation that by witholding certain sensitising foods from the lactating mother's diet the incidence of atopic symptoms is lowered, but not avoided.

These observations indicate the biological uniqueness of breast feeding that appears to be an exclusive way for the developing infant's immune system to be instructed and adapted to react through exposure to dietary antigens which, unlike all others, will never be encountered again in the course of life.

## References

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- <sup>2</sup> Raloff J. Bottle + breast = risky combination? *Science News* 1986; **130**:375.
- <sup>3</sup> Voglino GF, Ponzone A. Polymorphism in human casein. Nature 1972;238:149–50.

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## Congenital dislocation of the hip: to screen or not to screen

Sir,

The personal experience of Mr Dwyer<sup>1</sup> confirms my view that most abnormal hips may be safely and effectively diagnosed and treated at birth.<sup>2</sup> The excellence of his results reflects credit on him and on his team including the physiotherapists who I understand do the primary screening in his hospital.

In only one aspect does Mr Dwyer's practice differ appreciably from that recommended recently by the Standing Medical Advisory Committee and the Standing Nursing and Midwifery Advisory Committee.<sup>3</sup> The success of clinical screening in the neonatal period in his hospital seems to have suggested to him that follow up screening is not required beyond the age of three months. While this may be the case if screening is undertaken by 'a dedicated, experienced and skilful team', the availability of such support is difficult to achieve and maintain. For this reason the SMAC/SNMAC guidelines stated that 'it is essential to reassess the possibility of this condition until a child is seen to be walking normally'. Their report recommended that the hips of all children should be routinely surveyed not only at birth and on discharge from neonatal care, but also at 6 weeks, at 6-9 months, and between 15 and 21 months of age. This advice is surely sensible. Late cases of established congenital dislocation of the hip still occur even in the best centres and—as is well recognised—the later the diagnosis the more difficult the treatment and the more likely is the occurrence of avascular necrosis of the head of the femur.